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Exacerbation of Ventricular Arrhythmias During the Postoperative Period After Implantation of an Automatic Defibrillator

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The postoperative course of 68 consecutive patients treated with an implantable defibrillator during the period from 1982 through 1990 was studied. In 46 patients (group 1), no concomitant surgery was performed during the implantation. In 22 patients (group 2), concomitant surgery (coronary artery bypass [n = 12], valve replacement [n = 3] or arrhythmia surgery [n = 7]) was performed. All patients in group 1 were clinically stable before surgery, receiving an antiarrhythmic regimen chosen by serial drug testings. The same regimen was continued postoperatively.

Eight of the 46 patients in group 1 whose condition had been stable in the hospital for 19 ± 25 days preoperatively developed multiple episodes of sustained ventricular tachycardia 4 ± 9 days after implantation while receiving the same antiarrhythmic regimen. Although the exacerbation was transient in some patients, six required different antiarrhythmic therapy and one eventually died. Two additional patients had frequent and prolonged episodes of nonsustained ventricular tachycardia that could trigger the defibrillator, requiring changes in the antiarrhythmic regimen. Another patient had progressive cardiac failure and died on day 5. A marked (sevenfold) increase in asymptomatic ventricular arrhythmias was noted in 42% of the remaining 35 patients.

In group 2 (combined surgery), one patient developed refractory ventricular tachycardia 3 days postoperatively and died on that day. Three patients developed frequent nonsustained ventricular tachycardia postoperatively, requiring changes in the antiarrhythmic regimen. The overall surgical mortality rate was 4.4% (4.3% in group 1 and 4.5% in group 2) and was due to refractory ventricular tachycardia in two patients and cardiac failure in one.

Thus, during the postoperative period after defibrillator implantation, exacerbation of ventricular arrhythmias was common. The exacerbation was clinically significant in many patients and included multiple episodes of sustained ventricular tachycardia (with eventual death in some patients) or frequent prolonged nonsustained ventricular tachycardia that could trigger the defibrillator. A sevenfold asymptomatic increase in ventricular ectopic activity was noted in 42% of the remaining clinically stable patients. The long-term effect of the exacerbation is unknown.

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Implantable automatic defibrillators (1–8) have been used widely for the management of patients with malignant ventricular arrhythmias and their beneficial effect in preventing sudden cardiac death has been frequently reported (1–8). However, the mortality rate of the operation is not insignificant and infection frequently occurs (5,7, 9,10). In addition, several investigators (5,7,9,10) have noted intractable ventricular tachycardia after defibrillator implantation. This study was undertaken to investigate the frequency and nature of worsening of ventricular arrhythmia during the postoperative period after defibrillator implantation.

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Methods

Study patients. Sixty-eight consecutive patients who underwent implantation of an automatic defibrillator for malignant ventricular arrhythmias at Montefiore Hospital Medical Center between May 1, 1982 and December 31, 1990 were studied. Informed consent was obtained from all patients for implantation of the defibrillator. During the period when implantable defibrillators were investigational, the procedure was performed under the investigational protocol approved by the Institutional Research Review Committee in April 1982. The study was a retrospective analysis of prospectively obtained data in 34 patients during the period between May 1, 1982 and December 31, 1987 and prospective in 34 patients thereafter.

Forty-six patients (group 1) underwent defibrillator implantation without concomitant surgery and 22 patients (group 2) had concomitant surgery. Fourteen patients had concomitant coronary bypass surgery; two of these patients also had mitral valve replacement. Seven patients had concomitant surgery for arrhythmias such as subendocardial resection (n = 5) (11), cryosurgery (n = 1) (12) or septal myomectomy (n = 1) (13).

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One patient had mitral valve replacement. The 22 patients with concomitant surgery (group 2) were analyzed separately for the purpose of this study.

In group 1, 27 patients had sustained ventricular tachycardia and 19 had ventricular fibrillation. Sustained ventricular tachycardia was defined as lasting >30 s or requiring immediate termination by electric cardioversion because of a hemodynamic collapse. There were 35 men and 11 women, with a mean age of 58 ± 11 years. Coronary artery disease was present in 33 patients, dilated cardiomyopathy in 7, valvular heart disease in 4 and amyloid heart disease in 2. The mean left ventricular ejection fraction was 32 ± 15%.

In group 2, there were 16 men and 6 women, with a mean age of 62 ± 10 years. Eighteen patients had sustained ventricular tachycardia and four had ventricular fibrillation. Coronary artery disease was present in 19 patients (mitral regurgitation in 2), dilated cardiomyopathy in 1, valvular heart disease in 1 and hypertrophic cardiomyopathy in 1. The mean left ventricular ejection fraction was 37 ± 16%.

Antiarrhythmic therapy before the implantation. All 68 patients underwent serial drug testing by programmed stimulation and ambulatory electrocardiographic (ECG) monitoring (14–18). An antiarrhythmic regimen was selected on the basis of the results of serial testing. Although the regimens were considered ineffective by serial drug testing, all patients were in stable condition without spontaneous recurrence of symptomatic ventricular arrhythmias while receiving these antiarrhythmic regimens during the hospital stay before the defibrillator implantation.

Defibrillator therapy was not given to patients whose ventricular tachycardia induced by programmed stimulation became slower and better tolerated during drug therapy and whose spontaneous arrhythmias were reduced during ambulatory monitoring (15–18). Defibrillator therapy was also not given to patients whose frequent spontaneous recurrences of ventricular tachycardia could not be controlled by drug therapy.

Antiarrhythmic therapy during the postoperative period. All patients except those who had subendocardial resection received intravenous lidocaine or procainamide during the immediate postoperative period. The antiarrhythmic regimen chosen by preoperative drug testing was reintroduced when the patient was able to take medication by mouth. If a patient had recurrent sustained ventricular tachycardia while receiving intravenous medication postoperatively, administration of the oral antiarrhythmic drug chosen preoperatively was reinstituted. If a patient had recurrent ventricular tachycardia during the postoperative period while receiving the same oral antiarrhythmic regimen and had blood levels similar to preoperative levels, a different antiarrhythmic regimen was prescribed. Similarly, if a patient had frequent episodes of rapid nonsustained ventricular tachycardia postoperatively while receiving the regimen that was effective preoperatively, a different antiarrhythmic regimen was instituted to prevent triggering of the defibrillator by such arrhythmias (6). The decision to change the antiarrhythmic regimen in patients with frequent nonsustained ventricular tachycardia was not based on quantitative assessments by ambulatory ECG monitoring, but on findings on the telemetry monitor and clinical judgment of the investigators. In seven patients who had concomitant surgery for arrhythmias, antiarrhythmic drug therapy was chosen on the basis of postoperative drug testing.

Comparison of arrhythmia before and after operation. Ambulatory (ECG) monitoring and bedside telemetry were used to compare ventricular arrhythmias noted before and after operation. The preoperative ambulatory ECG recording obtained while a patient was receiving an antiarrhythmic regimen chosen by preoperative serial testing was compared with the postoperative recording obtained while the patient was receiving the same antiarrhythmic regimen.

Implantation of an automatic defibrillator. In group 1, all 46 patients underwent defibrillator implantation without concomitant surgery. The electrode-lead system used included a spring-patch configuration (6) in 6 patients, a patch-patch configuration (6) in 37 and a transvenous lead-subcutaneous patch configuration (Endotak) (19) in 3. A median sternotomy (6) was used in 39 patients, a lateral thoracotomy (6) was used in 3 patients, a subcostal approach (6) was used in 1 patient and transvenous and subcutaneous approach (for Endotak) (19) was used in 3 patients. In group 2, 14 patients had coronary bypass surgery (with mitral valve replacement in 2), 1 patient had mitral valve replacement, 5 patients had subendocardial resection, 1 patient had septal myomectomy and 1 had epicardial cryoablation during defibrillator implantation. The spring-patch configuration (6) was used in 2 patients and the patch-patch configuration in 20. All 22 patients had a median sternotomy.

Evaluation of postoperative ventricular arrhythmias in a control group. To assess effects of coronary bypass surgery on postoperative ventricular arrhythmias, 20 consecutive patients who underwent coronary bypass surgery without defibrillator implantation were studied by ambulatory ECG monitoring before and after operation. Patients with a recent (<2 weeks) myocardial infarction or left main coronary artery disease were excluded from the study.

Ambulatory ECG monitoring. The technique and accuracy of 24-h ambulatory ECG monitoring used in this study have been published (16). Nonsustained ventricular tachycardia was defined as three or more consecutive ventricular premature complexes at a rate ≥100 beats/min.

Statistical methods. Standard deviation (mean ± SD) was used as the index of dispersion of observed values. One-way analysis of variance (ANOVA), the Kruskal-Wallis test or chi-square test was used to compare appropriate variables between groups. A two-tailed p value <0.05 was considered significant.

Results

Drug therapy before operation (group 1). Antiarrhythmic therapy was given to 41 of the 46 patients before defibrillator

Table 1. Clinical Course of Eight Patients With Recurrent Sustained Ventricular Tachycardia After Defibrillator Implantation

Pt No.	Days Before Implantation*	Postimplantation	
		Time of Recurrence	Clinical Course
1	23	Day 3 to 13	Multiple recurrences over 10 days despite additional therapy; stabilized with amiodarone
2	40	Day 3 to 5	Multiple recurrences; stabilized with additional therapy
3	32	Day 19	Marked (30-fold) increase in NSVT in early postoperative period; additional therapy without effects with an eventual recurrence of VT on day 19; stabilized with amiodarone
4	30	Day 1 to 22	Multiple recurrences on various regimens with eventual death on day 22
5	16	Day 6 to 12	Multiple recurrences requiring additional therapy before eventual improvement; discharged on the same regimen
6	10	Day 4	Amiodarone therapy for VT for 5 months before the admission for syncope; although VT was inducible by PES, no spontaneous VT was noted before operation, amiodarone therapy was continued; there were multiple recurrences 4 days postoperatively.
7	14	Day 6	Frequent NSVT on day 5; numerous episodes of VT on day 6 requiring additional therapy; gradual improvement on increased dosage of sotalol
8	10	Day 3	Multiple VT (300 beats/min) on day 3 requiring additional therapy; frequent and fast (200 to 300 beats/min) NSVT during the next 10 days with gradual and partial improvement; discharged on the same therapy

*The number of days in the hospital before operation without clinical recurrence of sustained ventricular tachycardia (VT) in each patient. NSVT = episodes of nonsustained ventricular tachycardia; PES = programmed electric stimulation; Pt = patient; Time of Recurrence = time of recurrence of sustained ventricular tachycardia after operation.

implantation. In five patients, no antiarrhythmic agents were given because no therapy was considered beneficial by invasive or noninvasive evaluation of antiarrhythmic efficacy (12–16). A class 1A agent (procainamide, quinidine or disopyramide) was used in six patients, a class 1B agent (mexiletine or tocainide) in seven, a combination of class 1A and 1B agents in nine, a beta-adrenergic blocking agent in four, amiodarone in eight, a combination of a class 1A agent and a beta-blocker in five, sotalol in one patient and a combination of amiodarone and a class 1A agent in one.

Postoperative course (group 1) (Tables 1 and 2, Fig. 1). Eight of the 46 patients who had been in stable condition for 19 ± 25 days in the hospital developed sustained ventricular tachycardia 4 ± 9 days postoperatively. All patients except one (Patient 3) had multiple recurrences during the period (Table 1). Two patients (Patients 5 and 8) whose ventricular arrhythmia improved gradually were discharged from the hospital receiving the same antiarrhythmic regimen chosen before operation. In five patients, an additional or different antiarrhythmic agent (amiodarone in two patients) was used to stabilize the arrhythmia. One patient (Patient 4) died in the hospital as a result of refractory ventricular tachycardia. Antiarrhythmic regimen and blood levels before the implantation, blood levels of the same drugs after implantation at the time of recurrence and the final regimen at the time of discharge from the hospital are shown in Table 2.

In two other patients (Fig. 1), an additional antiarrhythmic agent (quinidine or tocainide, respectively) was used to suppress frequent nonsustained ventricular tachycardia noted postoperatively that could trigger the defibrillator. The frequency of nonsustained ventricular tachycardia by ambulatory ECG recording was not quantitatively assessed before the change in therapy. The decision was made by the investigators on the basis of the patients' clinical condition. After additional antiarrhythmic therapy, one patient no longer had nonsustained ventricular tachycardia. In the other patient, who was taking quinidine, the addition of tocainide markedly decreased the frequency of nonsustained ventricular tachycardia as judged by bedside telemetry monitoring. An ambulatory ECG recording obtained 7 days postoperatively while the patient was taking tocainide and

Table 2. Antiarrhythmic Regimens and Blood Levels in Eight Patients With Recurrent Ventricular Tachycardia After Defibrillator Implantation

Pt No.	Before Implantation		After Implantation	
	Regimen	Drug Level (mg/liter)	Drug Level* (mg/liter)	Final Regimen
1	PA	PA = 8, NAPA = 12.8	PA = 13, NAPA = 36	Amio
2	PA	PA = 10, NAPA = 8.5	PA = 12.5, NAPA = 14	PA + Q + Mx
3	N + Toc	N = 5	N = 3.3	Amio
4	PA	PA = 6.3, NAPA = 6.9	PA = 8.4, NAPA = 14.6	Died
5	Q + Mx	Q = 3.4	Q = 3.5	Q + Mx
6	Amio	Amio = 1.6, metabolite = 1.4	NA†	Amio + Q
7	Sot	NA	NA	Sot (increased)
8	PA + Mx	PA = 4.9, NAPA = 3.5	PA = 5.5, NAPA = 1.7	PA + Mx

*Drug level at the time of tachycardia recurrence postoperatively. In patients with multiple recurrences over multiple days, drug levels during the latest recurrence while the patient was taking the same drugs chosen preoperatively are shown. †This patient was treated with amiodarone (Amio) for 5 months before operation and amiodarone was continued postoperatively. Mx = mexiletine; N = disopyramide; NA = not available; NAPA = N-acetyl procainamide; PA = procainamide; Pt = patient; Q = quinidine; Sot = sotalol; Toc = tocainide.

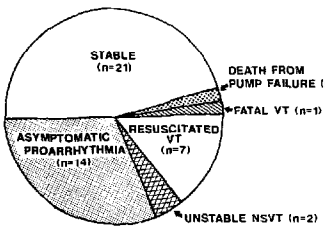

Figure 1. Clinical course of 46 patients in group 1 undergoing defibrillator implantation without a concomitant operation. ASYMPTOMATIC PROARRHYTHMIA = a marked increase (sevenfold) in ventricular premature complexes in clinically asymptomatic and stable patients; PUMP FAILURE = progressive cardiac failure with eventual death; STABLE = no significant increase in ventricular arrhythmias (two patients without postoperative ambulatory electrocardiographic recording were included); UNSTABLE NSVT = episodes of very frequent and rapid nonsustained ventricular tachycardia that might trigger the defibrillator and requiring additional antiarrhythmic therapy; VT = multiple recurrent sustained ventricular tachycardias postoperatively (see Table 1 for details of these eight patients).

quinidine revealed 114 runs of nonsustained ventricular tachycardia as compared with 35 runs preoperatively during administration of quinidine alone.

In summary, the antiarrhythmic regimen chosen before operation had to be changed during the postoperative period because of sustained ventricular tachycardia or frequent nonsustained ventricular tachycardia in 10 (22%) of the of 46 patients. In 2 of these 10 patients, the regimen chosen preoperatively could be reinstituted subsequently. One patient developed progressive cardiac failure postoperatively without recurrent ventricular tachycardia and died on day 5.

The remaining 35 patients were clinically stable postoperatively, receiving the antiarrhythmic regimen chosen preoperatively. Results of ambulatory ECG monitoring performed 9 ± 5 days after operation were compared with the monitoring results obtained preoperatively while the patients were receiving the same regimen. In two patients, postoperative ambulatory ECG recording was attempted but not obtained because of recorder malfunctions. Before operation, there was an average of 21.3 ± 42.1 ventricular premature complexes/h and 0.24 ± 0.56 episode of nonsustained ventricular tachycardia/24 h. Postoperatively, the average ventricular premature complexes/h increased to 74.5 ± 154.9 ($p < 0.05$) and the average nonsustained ventricular tachycardia/24 h increased to 1.8 ± 3.6 ($p < 0.02$). When individual patients were compared, a sevenfold increase in ventricular premature complexes (20) was noted in 14 (42%) of 33 patients who had matched ambulatory ECG recordings before and after operation while receiving the same antiarrhythmic regimen (Fig. 1). Nonsustained ventricular tachycardia was present in 6 of the 33 patients preoperatively and 15 postoperatively ($p < 0.02$). Antiarrhythmic drug levels drawn on the day of postoperative ambulatory ECG recordings were not significantly different from those of the preoperative period (quinidine 3.4 ± 1.1 before operation vs. 3.6 ± 1.8 mg/liter after operation; procainamide 7.6 ± 4.2 vs. 7.9 ± 5.3 mg/liter).

Postoperative clinical course (group 2). Twelve patients underwent coronary bypass graft surgery during defibrillator implantation. Two additional patients with coronary bypass surgery and mitral valve replacement were not included in this subgroup. Before operation, three patients were receiving a class 1A agent, three a class 1B agent, two both a class 1A and a class 1B agent and one a beta-blocker. Three patients did not receive any antiarrhythmic agent. Postoperatively, 1 of these 12 patients required additional antiarrhythmic therapy because of a marked increase in nonsustained ventricular tachycardias (8 runs of nonsustained ventricular tachycardia before vs. 1,356 runs after operation). Four of the remaining 11 patients had a sevenfold increase in ventricular premature complexes.

The remaining 10 patients in group 2 underwent a concomitant operation other than (or in addition to) coronary bypass grafting. Five patients had subendocardial resection, one a septal myomectomy, one epicardial cryosurgery for ventricular tachycardia and three mitral valve replacement or repair (two with coronary bypass surgery). Two patients with mitral valve replacement required an additional antiarrhythmic agent postoperatively for frequent episodes of rapid nonsustained ventricular tachycardia. One patient with dilated cardiomyopathy and recurrent well tolerated ventricular tachycardia underwent defibrillator implantation and cryosurgery for ventricular tachycardia originating from the epicardium. This patient developed multiple episodes of faster poorly tolerated ventricular tachycardia and ventricular fibrillation 3 days postoperatively and died on that day.

Postoperative course in the control group. Ambulatory ECG recordings were obtained before and after operation in 20 control patients who underwent coronary bypass surgery without defibrillator implantation. The average ventricular premature complex frequency was 4.15 ± 8.8/h before and 8.6 ± 17.6/h after operation ($p = 0.226$). The number of nonsustained ventricular tachycardias was 0 ± 0/24 h before and 0.3 ± 1.3/24 h postoperatively ($p = 0.333$). A sevenfold increase in ventricular premature complexes was noted in 2 of the 20 patients.

Surgical mortality. Three of the 68 patients died in the hospital within 30 days after operation for a surgical mortality rate (7) of 4.4%. This rate was 4.3% (2 of 46 patients) in group 1 and 4.5% (1 of 22 patients) in group 2. Two of the three deaths were due to refractory ventricular tachycardia and one was due to progressive cardiac failure.

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Discussion

The results of this study suggest that during the postoperative period after defibrillator implantation, 1) a clinically significant worsening of ventricular tachyarrhythmia defined as multiple recurrences of sustained ventricular tachycardia or prolonged rapid episodes of nonsustained ventricular tachycardia requiring changes in the antiarrhythmic regimen is noted in a significant number of patients, 2) the in-hospital mortality rate is not insignificant and is often due to the worsening of arrhythmias, and 3) a marked increase in the frequency of asymptomatic ventricular arrhythmias is noted in many other patients in clinically stable condition.

Comparison with other studies. The short-term results of our study are similar to those previously reported by others (7,9,10,21). Marchlinski et al. (10) also reported postoperative refractory ventricular tachycardia and one death. From data compiled by a manufacturer of defibrillators, Winkle and Thomas (7) reported that 40 (4%) of 949 patients died within 1 month after implantation (surgical mortality); 9 of the 40 who died had a sudden cardiac death during the postoperative period. Thirty-one of the 40 patients did not have concomitant surgery and 9 had concomitant surgery. Therefore, 31 (4.6%) of 669 patients without concomitant surgery and 9 (3.2%) of 280 patients with concomitant surgery died within 30 days postoperatively. It is unclear from their report (7) how many of the nine patients who died suddenly had concomitant surgery. In a study of 271 patients by Gohn et al. (21), the surgical mortality rate was 4% and 58.4% of surgical deaths were due to "arrhythmic complications." They (21) also reported that concomitant surgery during defibrillator implantation was not a determinant of surgical mortality. These investigators (7,10,21), however, did not report the total incidence of malignant ventricular arrhythmias, including ventricular tachycardia successfully treated in the hospital as reported in our study. In a study of 101 patients, Gartman et al. (9) reported that 11% of patients had sustained ventricular tachycardia postoperatively as compared with 13% in our study (9 of 68 patients; 8 in group 1 and 1 in group 2). Their surgical mortality rate was 4%. However, they did not report the incidence of worsening of nonsustained ventricular tachycardia necessitating changes in antiarrhythmic therapy. From our study and others (7,9,10,21), it appears that clinically significant worsening of arrhythmia is not uncommon after defibrillator implantation. Surgical mortality after implantation is frequently due to the aggravation of ventricular arrhythmia or the appearance of incessant ventricular tachycardia or ventricular fibrillation.

Our study also reports a marked (sevenfold) increase in asymptomatic ventricular premature complexes (20) in the remaining clinically stable patients. Although the clinical significance of the asymptomatic increase in ventricular arrhythmias is unknown, no other studies have been done to quantitatively compare ventricular arrhythmias noted before and after operation.

Mechanisms of the arrhythmogenesis. This study suggests that implantation of currently available defibrillator and electrode lead systems or the operation associated with implantation may aggravate ventricular arrhythmias in some patients. The cause of this aggravation is unknown. If it is related to mechanical irritation or inflammation after the procedure, future generations of defibrillators using subcutaneous patch electrodes may not be associated with such problems.

Metabolic and electrolyte changes during the immediate postoperative period may have played a role in the worsening of arrhythmias in some patients, although in many patients the recurrence of sustained ventricular tachycardia was noted several days to weeks after the operation with adequate blood levels of antiarrhythmic drugs and normal serum electrolytes (Tables 1 and 2). In addition, ambulatory ECG monitoring performed 9 ± 5 days after surgery in clinically stable patients showed a significant increase in ventricular ectopic activity, further suggesting that the worsening may not be solely due to postoperative transient metabolic and electrolyte changes. However, it is possible that serum electrolyte changes, ischemia or pericarditis may have played some role in the arrhythmogenesis. Atrial arrhythmias commonly associated with postoperative pericarditis were frequently noted in our patients. However, no correlation between atrial arrhythmias and the worsening of ventricular arrhythmia could be established in this study. It is unclear why in most patients, the onset of sustained arrhythmias was noted several days postoperatively (Table 1).

One may argue that a concomitant operation such as coronary bypass grafting may be responsible for the worsening. However, no patient in group 1 had a concomitant operation. In addition, although the 20 patients who had coronary bypass grafting without defibrillator implantation also had a slight increase in ventricular arrhythmias postoperatively, the increase was not as significant as that noted in the group 1 patients or in the 12 patients who had both defibrillator implantation and coronary bypass grafting. Regardless of the mechanism of the worsening of arrhythmias, the worsening is clinically significant in many patients and in some leads to in-hospital death during the postoperative period as shown in this study and others (7,10,21).

It is also unknown if the worsening of arrhythmia noted during the short-term follow-up period has any long-term effects. Further studies are necessary to determine the mechanism of the aggravation of arrhythmia and the long-term prognostic significance.

Study protocol. The study patients were classified into two groups and analyzed separately to exclude possible effects of concomitant surgery on the postoperative course. However, the incidence of clinically significant worsening of arrhythmias was similar in the two groups. Such worsening was defined as recurrence of ventricular tachycardia or frequent nonsustained ventricular tachycardia that required an additional antiarrhythmic agent in patients who had been

in stable condition preoperatively. Eight patients with a recurrence had been in stable condition without ventricular tachycardia for ≥19 ± 25 days (range 12 to 60) before operation and had multiple recurrences 4 ± 9 days postoperatively ($p < 0.05$) (Table 1).

Worsening of arrhythmia noted after an implantation of a defibrillator was defined by ambulatory ECG monitoring in the remaining 35 patients. The clinical significance of a marked (sevenfold) increase in the frequency of asymptomatic ventricular arrhythmias noted during ambulatory monitoring is unclear. However, the increase noted postoperatively is unlikely to be due to the day to day variations in the frequency of ventricular premature complexes in view of the stringent criterion used in this study that was proposed by other investigators (20) to exclude such possibility of day to day variations.

Limitations of the study. The relatively small number of patients in group 1 is a limitation. However, no other investigation has addressed the issue systematically and further studies in a larger group of patients will be needed to confirm or repudiate our findings. Our study does not address the long-term effects of the worsening of ventricular arrhythmias noted. Although the worsening appeared to be transient and the previously chosen antiarrhythmic regimen could be reinstituted in some patients, most of the patients received an additional or a different agent postoperatively, making it difficult to determine if the worsening was transient. Additional studies to answer this question may be difficult to conduct because the outcome of patients treated with defibrillator implantation needs to be compared with that of similar patients treated with similar drugs without a defibrillator.

Clinical implications. The results of this study do not negate the value of the automatic implantable defibrillator in the management of patients with malignant arrhythmias. Additional studies may identify the mechanism for the worsening of the arrhythmias and correct the problem. However, when choosing a patient for defibrillator therapy and interpreting the results of clinical follow-up of patients treated with an implantable defibrillator, it should be borne in mind that the current technology of implantation may change the clinical course of patients. The surgical mortality rate is not insignificant (7,9,21) and is frequently due to the development of incessant ventricular tachycardia during the postoperative period (7,10,21). In addition, it is possible that the exacerbation of arrhythmia noted during this period may have long-term effects. Therefore, the rate of recurrence of malignant arrhythmias in patients with an implantable defibrillator may not reflect the true rate of recurrence of malignant arrhythmias in those patients treated with the same medical regimen without a defibrillator.

Because of the many reports of very low rates of sudden death in patients treated with a defibrillator, some investigators (22,23) have suggested that all patients with ventricular tachycardia or ventricular fibrillation should be treated with an implantable defibrillator even when an effective regimen by programmed stimulation criteria could be identified. Their argument may be based on the fact that reported sudden death rates in patients treated with an implantable defibrillator are often lower than those in patients treated with antiarrhythmic drugs that prevented induction of ventricular tachycardia by programmed stimulation (24). Such argument suggesting obsolescence of electrophysiologic studies may not be valid (25), not only because the sudden death rate overestimates the benefits of defibrillator therapy by not including the surgical mortality and arrhythmia-related nonsudden deaths (26,27), but also because defibrillator therapy may alter the natural course of the disease process in some patients by aggravating the arrhythmias as addressed in this study.

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